

A Comment on the Relationship of Recent Research on CSF Production and Reabsorption to the Pressurestat Model

There have been a few research articles about CSF lately. Some people felt this would totally destroy the whole pressurestat model used in Upledger CST. However, Upledger Instructor Tim Hutton explains how this actually still supports the Upledger pressurestat model.

Recently, a lot of very interesting research has come out on how cerebrospinal fluid (CSF) is produced and reabsorbed in the central nervous system (CNS). This research has totally transformed our understanding of this neurological process. I wanted to comment briefly on the relationship of this recent research to the pressurestat model that is taught in Upledger CranioSacral Therapy classes.

The pressurestat model is a model that was developed by John **Upledger** and Ernest Retzlaff to explain the rhythmic expansion and contraction of the cranial vault and the concurrent external and internal rotation of the tissue of the body known as the craniosacral rhythm (CSR).

The classic neurological model for CSF production and reabsorption, which has been taught for years, states that all CSF production occurs in the choroid plexi, particularly those in the two lateral ventricles, and all CSF reabsorption takes place in the arachnoid villi of the venous sinus system. Recent research has demonstrated, however, that a significant portion of CSF production and reabsorption, perhaps as much as 60 to 70 percent, occurs locally at the blood brain barrier in the capillary beds of the central nervous system (CNS). (See for example the review article, Chikly, B., Quaghebeur, J., Reassessing cerebrospinal fluid (CSF) hydrodynamics: A literature review presenting a novel hypothesis for CSF physiology, *Journal of Bodywork & Movement Therapies* (2013), Online publication complete: 12-APR-2013). Only 30 to 40 percent of the total CSF production seems to occur in the choroid plexi. Recent research has also shown that a significant portion of the global CSF production is reabsorbed directly into the lymphatic system rather than through the arachnoid villi into the venous sinuses.

Upledger and Retzlaff's pressurestat model proposed that the CSR was the result of minute rhythmic pressure changes in the cranial vault that occur as a consequence of the action of the body's mechanism for controlling baseline CSF pressure. They postulated that while CSF reabsorption was constant, CSF production in the choroid plexi cycled on and off, at a rate of 6 to 12 cycles per minute, in response to signals from pressure and stretch receptors located primarily in the sagittal suture.

Given that we now know that only a portion of CSF production takes place in the choroid plexi, does this new research have any implications with regard to the pressurestat model?

It is first important to note that the pressurestat model and the classical neurological model of CSF production and reabsorption are two separate and distinct things. In my experience, students often confuse the two. The pressurestat model does say anything about where or how the CSF is produced or reabsorbed. It merely states that the production is cyclic and the reabsorption is constant. Thus the pressurestat model is not fundamentally dependent on the validity of the classical neurological model. Changing the location or mechanism of fluid production and reabsorption does not necessarily impact the pressurestat model. In particular, the fact that a large proportion of CSF may be reabsorbed somewhere other than the arachnoid villi is irrelevant to the pressurestat model, so long as what reabsorption there is remains relatively constant.

It is my contention, speaking as a scientist, and as someone who has been doing CranioSacral Therapy (CST) for many years, that this new research does not conflict in any way with the pressurestat model, and in fact explains some things that have puzzled me for a long time. The two fit together perfectly. I would, in fact, be very surprised if the body did not have both a mechanism for local production and reabsorption of CSF, and one for global production and reabsorption as well. These two mechanisms serve very different purposes in the CNS.

I have always been vaguely uneasy with the classical neurological model, with the idea that all CSF production takes place at the plexi and all reabsorption takes place in the villi. CSF plays the roles of both interstitial fluid and lymph in the CNS. It carries away metabolic waste from the tissue. The presence of excess metabolic waste in the interstitial space is highly inflammatory and it would seem particularly important for the proper functioning of the body to prevent such accumulation in the tissue of the CNS. It made no sense to me that the body would rely on a mechanism for waste removal that was very slow, which turned the fluid volume over only a few times a day, and one that required the waste products to be transported long distances through CNS tissue before they were removed. Local production and reabsorption of CSF would be much more efficient, and would allow for nearly instantaneous removal of waste products into the venous blood flow. Accomplishing this removal locally also means that waste products do not need to be transported through the CNS tissue to be removed, potentially producing more inflammation along the way.

It is difficult, however, for me to see how the body would be able to control baseline CSF pressure if all the fluid production was local. How would the body coordinate billions of local CSF production centers? There needs to be a mechanical sensor that provides the signal to

indicate when CSF pressure is too high or too low. Distributing that signal to billions of local CSF production centers would seem to be a daunting task. The body is generally very efficient, doing things in the easiest possible way, and Ockham's Razor would argue for a much simpler arrangement. Stretch and pressure receptors in the sutures seem to me to be a simple and ideal way to provide the mechanical signal needed to control fluid pressure, and so long as a significant portion of total CSF production takes place in a centralized location, passing the signal from the sutures to that location would be an elegant solution to this problem.

As mentioned above, the essence of the pressurestat model is that, while the fluid reabsorption is constant, the fluid production cycles on and off. Where the fluid is reabsorbed is irrelevant as far as this model is concerned, so it makes no difference to the pressurestat if the globally produced CSF is reabsorbed into the lymph, locally in the region of the choroid plexi where it is produced, or into the venous sinuses via the arachnoid villi, or any combination thereof. The pressurestat model also does not require that all the fluid production be turned on and off in this way. So long as a large enough percentage of total production cycles on and off, it would be possible to control baseline CSF pressure effectively. The recent research still postulates 30 to 40 percent of total CSF production taking place in the choroid plexi, presumably more than enough to run the pressurestat.

As evidence that the mechanical signal needed to control baseline CSF pressure does indeed come from the sagittal suture, I offer the following case report. Several years ago I treated a young man, age 14, who a few years previously had taken a fall on his bicycle and landed on his head, crushing the helmet he was wearing over the left parietal. Sometime after the accident he developed a severe constant category 9 - 10 headache. Medical examination revealed that he had severely elevated CSF pressure and he was given a diagnosis of pseudotumor cerebri. When I initially examined him, he exhibited no CSR anywhere on his body. He was not in a stillpoint or a significance detector stop. It felt more like the CS system simply did not exist, as if the master switch had been turned off. Examination also revealed a severely compressed left parietal, totally jamming the sagittal suture. Releasing the left parietal immediately restored good CSR throughout his tissue, and two weeks after the treatment, once the accumulated metabolic waste had been carted off and the resulting inflammation had died down, his headache went away.

This one treatment convinced me of the basic validity of the pressurestat model. With the sagittal suture jammed, stretch sensors in the suture never fired, and were unable to send a signal to the choroid plexi instructing them to turn off fluid production. In addition, pressure sensors in the suture were constantly being stimulated, in effect telling the body to produce more fluid. The plexi continued to produce fluid until the baseline CSF pressure matched baseline arterial pressure, at which point it was no longer possible to filter CSF from the arterial blood. The entire system shut down and the result was a massive accumulation of waste products in the CNS

and a horrendous headache. Releasing the suture allowed the system to begin functioning again and start carting off the metabolic waste.

All scientific models explain certain behaviors of the system they are meant to describe, and do not explain others. Thus, all scientific models have limited regions of validity. Another way to say this is to note that all scientific models are to some extent oversimplifications. Hopefully they are useful oversimplifications, but they are oversimplifications nonetheless. I am certain the pressurestat model is an oversimplification of what happens in the tissue, but I am also convinced that it offers a good explanation of the basic physiological basis for the CSR.

In what areas is it likely that the pressurestat model is valid, and in what areas is it likely not valid? The pressurestat model explains the physiological basis for the CSR quite well, and is consistent with the fact that the body can go into stillpoint. Presumably stillpoint is a process mediated by the CNS to release tensions in the autonomic nervous system, and in the body as a whole. I see no theoretical conflicts between this behavior and the pressurestat model. The pressurestat model does not, however, explain such behavior as the significance detector. In particular, it does not explain the fact that one can dialogue, silently or out loud, with the CSR, and it will turn on and off locally in response to yes/no questions from the therapist. These behaviors are much better explained using a model that considers the CSR to be a vehicle for communication between the conscious mind of the therapist and the nonconscious of the patient.

It is interesting to note that any rhythm in the body can be used to dialogue with the nonconscious of the patient in this way. This includes visceral motility, any of the lymphatic rhythms, or indeed any of the many other rhythms of the body. In my experience, this seems to be a universal property of the body/psyche, not a property of any individual rhythm. Asking yes/no questions of any of these rhythms will cause the rhythm to turn on and off locally, provided the therapist has set that intention, in effect provided the therapist has made that agreement with the Inner Physician of the patient.

In summary, I see no conflict whatsoever between this latest research and the pressurestat model. This research, in fact, clears up some troubling aspects of the classical neurological model. Now that we understand how it all works, I would be surprised if the body functioned any other way.

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